442
EARLY STRUCTURAL HIP ABNORMALITIES ARE ASSOCIATED WITH OBESITY AND BODY COMPOSITION MEASURES – A 3.0T MRI COMMUNITY-BASED STUDY

A. Teichtahl †, Y. Wang ‡, S. Smith ‡, A. Wluka ‡, D. Urquhart ‡, G. Giles §, S. Hussain ‡, F. Cicuttini ‡. † *Baker IDI/Monash Univ., Melbourne, Australia;* ‡ *Monash Univ., Melbourne, Australia;* § *Cancer Council Victoria, Melbourne, Australia*

Purpose: Although obesity is a risk factor for hip osteoarthritis (OA), the role of body composition, if any, is unclear. This study examines whether obesity and body composition are associated with hip cartilage changes using magnetic resonance imaging (MRI) in community-based adults.
Methods: 141 community-based participants with no clinical hip disease, including OA, had BMI and body composition (fat mass and fat free mass) measured at baseline (1990 to 1994), and BMI measured and 3.0T MRI performed at follow-up (2009-2010). Femoral head cartilage volume was measured and femoral head cartilage defects were scored in the different hip regions.
Results: For females, baseline BMI (β=-26mm^3, 95% CI -47 to -6mm^3, p=0.01) and fat mass (β= -11mm^3, 95% CI -21 to -1mm^3, p=0.03) were negatively associated with femoral head cartilage volume. Also, while increased baseline fat mass was associated with an increased risk of cartilage defects in the central superolateral region of the femoral head (OR= 1.08, 95% CI 1.00-1.15, p=0.04), increased baseline fat free mass was associated with a reduced risk of cartilage defects in this region (OR=0.82, 95% CI 0.67-0.99; p=0.04). For males, baseline fat free mass was associated with increased femoral head cartilage volume (β= 40mm^3, 95% CI 6 to 74mm^3, p=0.02)
Conclusions: Increased fat mass was associated with adverse hip cartilage changes for females, while increased fat free mass was associated with beneficial cartilage changes for both genders. Further work is required to determine whether modifying obesity and in particular body composition alters the development of hip OA.

443
VASTUS MEDIALIS FAT INFILTRATION – A MODIFIABLE DETERMINANT OF KNEE CARTILAGE LOSS

A. Teichtahl †, A. Wluka ‡, Y. Wang ‡, P. Wijethilake ‡, B. Strauss ‡, J. Proietto §, J. Dixon ‖, G. Jones ¶, A. Forbes ‡, F. Cicuttini ‡. † *Baker IDI/Monash Univ., Melbourne, Australia;* ‡ *Monash Univ., Melbourne, Australia;* § *Austin Hosp. and Univ. of Melbourne, Melbourne, Australia;* ‖ *Baker IDI, Melbourne, Australia;* ¶ *Menzies Res. Inst., Tasmania, Australia*

Purpose: There is growing interest in the role of intramuscular fat and how it may influence clinical outcomes. Vastus medialis (VM) is a functionally important quadriceps muscle that helps to stabilise the knee joint. This longitudinal study examined the determinants of VM fat infiltration and whether VM fat infiltration influenced knee cartilage volume.
Methods: 250 participants without any diagnosed arthropathy were assessed at baseline between 2005 and 2008 and at follow-up between 2008 and 2010. Ambulatory and sporting activity were assessed and magnetic resonance imaging was used to determine knee cartilage volume and VM fat infiltration.
Results: Age, female gender, BMI and weight were positively associated with baseline VM fat infiltration ($p \leq 0.03$), while ambulatory and sporting activity were negatively associated with VM fat infiltration ($p \leq 0.05$). After adjusting for confounders, a reduction in VM fat infiltration was associated with a reduced annual loss of medial tibial (β = -10mm^3; 95% CI -19 to 0 mm^3; $p = 0.04$) and patella (β = -18mm^3; 95% CI -36 to 0 mm^3; $p = 0.04$) cartilage volume.
Conclusions: This community-based study of healthy adults has shown that VM fat infiltration can be modified by lifestyle factors including weight loss and exercise, and reducing fat infiltration in VM has beneficial effect on knee cartilage preservation. The findings suggest that modifying VM fat infiltration via lifestyle interventions may have the potential to reduce the risk of knee OA.

444
SYSTEMATIZING MURINE MENISCAL AND LIGAMENT MODIFICATIONS DURING OSTEOARTHRITIS

B. Poulet †, G. Bou-Gharios ‡, A.A. Pitsillides §. † *UCL, London, United Kingdom;* ‡ *Univ. of Liverpool, Liverpool, United Kingdom;* § *Royal Vet. Coll., London, United Kingdom*

Purpose: Osteoarthritis (OA) is a disease that affects all joint tissues, and changes have been described in the articular cartilage (AC), subchondral bone and synovium. Pathologies in menisci and ligaments, however, are rarely analysed, although both are known to play vital roles in the mechanical stability of the joint. Our aim is to describe histological modifications of the meniscus and collateral and cruciate ligaments in various models of OA, including during spontaneous OA in the Str/ort mouse, in post-traumatic OA in the chronic destabilization of the medial meniscus (DMM) model and our non-invasive knee joint trauma loading model.
Methods: Histological sections from mouse knee joints of i) Str/ort mice at different ages (8, 18, 26 and 40weeks), ii) C57Bl/6 mice 8 weeks after DMM surgery, and iii) CBA mice 5 weeks following mechanical trauma, stained with Toluidine Blue or Safranin O were used. Meniscal and ligament pathologies were assessed for pathophysiological changes.
Results: The extent of meniscal pathological changes was associated with the severity of AC lesions in the Str/ort mouse. Mild meniscal changes were seen from grade 3 AC lesions, with osteophyte formation at the tip of the meniscus and new ossification in the fibrocartilagenous outer part of the meniscus. Joints with grade 4 AC lesions also showed pathological changes in the attachment of the meniscus to the joint capsule, including hyperplasia and cell hypertrophy. In Str/ort mouse joints showing complete loss of AC (grade 6), the meniscus was eroded. The medial meniscus of DMM-induced and the lateral meniscus of trauma-induced OA joints showed similar changes (Table 1). Ligaments in all three models showed changes that were consistent with endochondral ossification, including chondrogenic changes such as increased extracellular matrix (ECM) staining, loss of fibre alignment and cell hypertrophy and clustering near ligament insertion sites. Ossification, with small areas of bone formation with marrow cavities evident, was seen in the collateral and transverse meniscal, but never in the cruciate ligaments.
Conclusions: Modifications in menisci and ligaments follow overt AC degeneration in murine OA, at least in the Str/ort mouse where such changes appear after the first signs of AC degradation. Although the aetiology and the consequences of such changes remain unknown, they will influence stability and load transmission of the joint, and may therefore contribute to OA progression. Description of such tissue changes in studies involving mouse models in addition to AC degradation are important to understand the pathogenesis of OA and the potential effects of specific targets for therapy. In addition, these changes may have important roles in restricting range of movement and pain, which represent major human clinical symptoms of OA.

Table 1
Summary of meniscal and cruciate and collateral ligaments pathological changes in three models of murine OA. (Grades for the Str/ort mouse model correspond to AC lesion severity grades, as defined by the OARSI grading system).

	Natural model (Str/ort)	Trauma model	Surgical model (DMM)
Meniscus			
Meniscal osteophyte	*Grade 3-5*		*Yes*
Outer part Chondrogenesis	*Grade 4*	*Yes*	*Yes*
Outer part Ossification	*Grade 3-6*	*Yes*	*Yes*
Attachment hypertrophy	*Grade 4-6*		*Yes*
Erosion	*Grade 6**		*Yes**
Cruciate ligament			
Strong ECM staining	*Grade 3-6*	*Yes*	*Yes*
Cell hypertrophy	*Grade 3-6*	*Yes*	*Yes*
Cell clustering	*Grades 4-6*	*Yes*	*Yes*
Loss of alignment	*Grade 3-4*	*Yes*	*Yes*
Collateral ligament			
Chondroplasia	*Grade 4-5*		
Ossification	*Grade 4-5*		

* when grade 6 AC degradation.

445
A LARGE INFRAPATELLAR FAT PAD PROTECTS AGAINST KNEE PAIN AND LATERAL TIBIAL CARTILAGE VOLUME LOSS

A. Teichtahl †, E. Wulidasari ‡, S. Brady ‡, Y. Wang ‡, A. Wluka ‡, C. Ding ‡, G. Giles §, F. Cicuttini ‡. † *Baker IDI/Monash Univ., Melbourne, Australia;* ‡ *Monash Univ., Melbourne, Australia;* § *Cancer Council Victoria, Melbourne, Australia*